Abstract

Mice that were genetically susceptible to developing atherosclerosis were fed a normal chow diet and were exposed to about 160 μg/m^3 fine particle air pollution ($PM_{2.5}$) for 4 days per week, 6 hours per day for 6 months in Riverside, CA. Control mice were exposed to purified air. The goal of this study was to determine if exposure to pollutant particles would accelerate the development of atherosclerosis in these mice and if the exposures also caused increased evidence of heart dysfunction. The development of atherosclerotic plaque was followed during the experiment using a non-invasive ultrasonic microscope method and a subset of mice was equipped with a telemetry device that relayed their electrocardiograms to a computer. We examined several factors relevant to mechanisms of development of atherosclerosis and heart disease. At the end of the six month exposure we measured the development of plaque in the aorta of each mouse and also measured several biomarkers in serum that are known to be elevated in humans with coronary artery disease. After six months of exposure to elevated levels of $PM_{2.5}$ there was, on average, a 60% increase in the amount of plaque in the arteries of these mice than there was in the air-exposed mice. Although there was substantial animal to animal variation, this finding was significant ($p \leq 0.05$). The level of C-reactive protein, which is one indicator of systemic inflammation, was positively associated with the development of plaque in the CAPs-exposed mice, but not in the air-exposed mice. The conclusion drawn from this study is that exposure to particulate pollution ($PM_{2.5}$) in California significantly accelerates atherosclerosis development.